

Endovascular Treatment for Intracranial Atherosclerotic Stenosis

Pitfalls and Problems

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Summary

Forty-five cases of intracranial atherosclerotic stenoses greater than 70% for intradural lesions or 60% for extradural lesions were treated by PTA or stenting. The stenotic lesions were successfully dilated in 44/45 patients and future stroke was prevented during a mean 29 month follow-up period. Stenotic ratio significantly reduced in stent-treated group compared with PTA-treated group and stenting was effective for cases refractory to PTA, such as elastic recoil or restenosis. However, stenting had its own drawbacks, such as difficulty in delivery, migration, and acute thrombosis.

Introduction

Endovascular treatment for intracranial atherosclerotic stenoses has become popular with the recent development of PTA balloon catheters for intracranial use and flexible coronary stents^{1,2,4,5,6,7,8,9}. Several papers reported the effectiveness of endovascular treatment of intracranial arterial stenosis^{1,2,4,5,6,7,8} but its pitfalls and unsolved problems such as perforating

artery occlusion related to PTA/stenting have been still unknown⁹. We report our results and pitfalls related to procedures in this paper.

Material and Methods

Forty-five cases of intracranial atherosclerotic stenoses were treated with PTA or stenting. Twenty-eight cases were intracranial internal carotid stenoses, seven middle cerebral arterial stenoses, ten vertebro-basilar stenoses. Thirty-five cases were treated with PTA alone and ten cases were treated with stenting. Our indication of the treatment was symptomatic stenosis greater than 70% for intracranial intradural lesions and stenosis greater than 60% for intracranial extradural lesions. Stenting was performed for unsuccessful PTA cases, such as elastic recoil, dissection, or restenosis. As for PTA, smaller balloon than normal diameter was used and inflated slowly. A single lumen PTA balloon catheter (Stealth, Boston Scientific) or a double lumen PTA balloon catheter for coronary artery was used for PTA. A PTA balloon was inflated for 1 to 2 minutes with 6 to 12



Figure 1 CT findings. Bilateral occipital low density areas were demonstrated.

atm. All cases but one were approached via transfemoral route. In one case of basilar stenosis, stenting was performed through the surgically exposed C1 vertebral artery. As for stents, balloon expandable stents such as GFX, S-670 (Medtronic), NIR (Boston) were used for cases refractory to PTA. Antiplatelet drug (ticlopidine 200 mg/day) was administered at least two weeks before treatment and continued after treatment. All procedures were performed under systemic heparinization which continued for several days in stent-treated cases.

Follow-up angiography was performed 3 to 6 months after treatment.

Results

Forty-four cases were successfully dilated with PTA or stenting. In one case, a PTA balloon catheter was not introduced into the lesion because of the tortuous ICA. The stenosis improved from 83% to 28% in PTA group, while 78% to 7% in stent group. Stent-treated cases demonstrated greater initial gain compared with PTA group. Restenosis rate in PTA or stent-treated group was 25% and 12.5%, respectively. As for complication, in PTA group, one case showed fatal hyperperfusion hemor-

rhage 30 minutes after successful PTA and died. The other case caused subarachnoid hemorrhage 6 hours after PTA supposed from the associated aneurysm. One case caused acute occlusion by a guidewire but successfully recanalized without new neurological deficits. One case caused minor neurological deficit after PTA for the basilar artery due to supposed perforating artery occlusion. In stent group, there was one stent migration, which needed stent retrieval by a snare wire. One acute stent thrombosis occurred, which was successfully recanalized but minor deficit remained. Ten of two stents were not successfully delivered to the target lesion due to the tortuosity of the vessel. One case approached from the vertebral artery at the C6 level caused a vertebral arterio-venous fistula, which was treated transvenous coil embolization.

The mortality rate was 1/45 (2.2%) and morbidity rate was 3/45 (6.7%) including one major morbidity. The morbi-mortality rate was 8.1% in PTA group, and 12.5% in stent group.

Three patients caused lesion non-related ischemic stroke but lesion related ischemic stroke did not occur in all cases during mean 29-months follow-up period.

Representative case

A 69-year-old male became delirious with disturbance of vision. CT scan demonstrated bilateral occipital lobe infarction and angiography demonstrated severe basilar artery stenosis with the faint posterior communication artery (figure 1,2). PTA was performed for the basilar stenosis but failed to dilate the artery due to elastic recoil. Then a 3x12 mm NIR stent was delivered and the basilar artery was dilated via the surgically exposed vertebral artery between the C1 and occipital bone (figure 3).

Discussion

Natural course of the intracranial arterial stenosis is unknown, but according to Craig, stroke rate is reported nearly 8%/year³. In our series, no patient caused lesion-related stroke in 29 months follow-up period, although 3 patients had lesion non-related stroke and 8.9% of perioperative morbi/mortality was encountered. From our retrospective study, we suppose endovascular therapy was effective for



Figure 2 Vertebral angiography before treatment A) A-P view, B) Lateral view. Severe stenosis was demonstrated in the basilar artery. Arrow shows the tortuous bend at the C2 level.

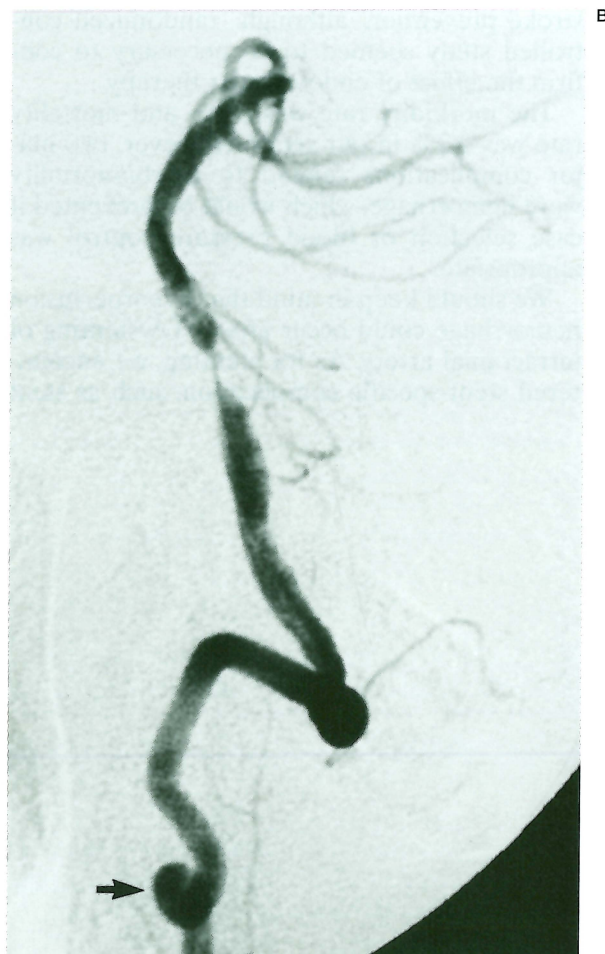
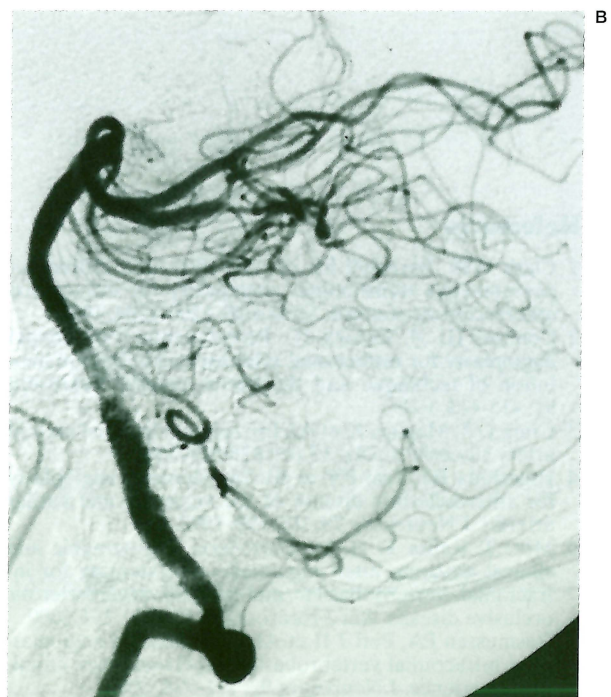


Figure 3 Vertebral angiography after stenting A) A-P view, B) Lateral view. Stenotic lesion was well dilated with stenting.



stroke prevention, although randomized controlled study seemed to be necessary to confirm the effect of endovascular therapy.

The morbidity rate was 6.7% and mortality rate was 2.2% in our series. However, two major complications related to morbi/mortality were hemorrhage, which would be prevented if case selection or blood pressure control was appropriate.

We should keep in mind that hyperperfusion hemorrhage could occur after PTA/stenting of intracranial artery. As for stenting, we encountered stent-specific complication, such as stent

thrombosis, stent migration, although stent was very effective device for lesions refractory to PTA and brought significant initial gain^{5,6,9}. Strict control of ACT and manipulation of microcatheter and guidewire under fluoroscopic control are necessary.

Conclusions

Endovascular therapy for the intracranial arterial stenoses was effective for stroke prevention but special care should be taken to reduce the perioperative complications.

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